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The Practical Management of Dizziness

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• *The problem of dizziness is greatly simplified if: (1) A definite routine is followed in the history taking and the steps of the examination; (2) the examination is done in two stages; and (3) a simple classification is used in cases of dizziness of vestibular origin whereby three groups are distinguished on the basis of localization of the origin. The etiologic diagnosis is thereby narrowed down to a relatively few possibilities.*

The treatment consists of measures to relieve the symptoms and to reverse the underlying disease which produced the symptoms.

DIZZINESS is one of the common complaints to be heard in a physician's office. It may occur in a variety of systemic diseases as well as in the more localized disturbances of the inner ear.

The management of the patient with dizziness often presents a somewhat confusing and time-consuming problem. The problem arises mainly because of the broad field to be covered in differential diagnosis and the frequent lack of definite clues to indicate the cause.

The first objective in dealing with this complaint is to determine, if possible, whether the dizziness is true vertigo arising from the vestibular system in the ear and its central nervous connections, or whether it arises from some other system of the body which is concerned with equilibrium.

Ocular disturbances, especially if they are of recent origin, may affect equilibrium, particularly during motion, and cause a complaint of confusion or lightheadedness. They do not cause a sense of rotation or turning such as occurs in vertigo of vestibular origin.

The body depends also on kinesthetic sense for maintenance of position and equilibrium. A disturbance of this sense in such diseases as locomotor ataxia or syringomyelia may cause impairment of equilibrium during motion.

Diseases of the circulatory system are more frequently a source of a complaint that may be confused with true dizziness or vertigo. Persons who are subject to attacks of syncope may use the term "dizzy spell" or "blacking out." Certain syndromes such as the carotid sinus syndrome and Stokes-Adams syndrome may cause the complaint of "dizziness." These circulatory disturbances are not likely to cause a true sense of rotation or movement. If there has been a definite loss of consciousness lasting at least for a few moments, the attack is probably not due to a vestibular disturbance but the origin is likely to be found in the circulatory system or more rarely in the cerebral cortex.

Dizziness is a subjective sensation and may be met with in persons with an unstable psychic make-up. There is real doubt as to whether vestibular vertigo with its accompanying nystagmus, ataxia and vegetative symptoms can be caused entirely by psychic disturbance. There has been ample evidence that Meniere's disease or hydrops of the labyrinth

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tends to be aggravated by psychic stress, but, in the absence of such a disease entity, it is doubtful if true vestibular vertigo can occur purely on a psychogenic basis. Psychogenic dizziness will not be described as a clear-cut sense of movement such as occurs in labyrinthine vertigo, nor will the patient give a description of a postural vertigo such as is frequent in organic vestibular disturbances.

DIAGNOSIS

The history obtained from the patient is the most important step in the diagnosis of dizziness. It must include specific questioning to bring out all the characteristics of the dizzy spell and to indicate whether vertigo occurs only when certain positions are assumed or certain changes in position made. The history must also include questioning regarding possible related signs and symptoms.

Much of the confusion connected with the management of vertigo can be avoided by the use of a definite routine of examination and by using a simple classification as a basis for diagnosis.

A routine method of examination that serves well in cases of dizziness is as follows:

1. Routine ear, nose and throat examination. Function of cranial nerves. Tests for incoordination, Rombergism, past pointing, etc.
2. Hearing tests.
3. Spontaneous nystagmus.
4. Positional nystagmus.
5. Caloric tests of labyrinthine function.
6. Routine laboratory work including urinalysis and blood counts. Blood pressure.

It has been found advisable to divide the examination into two sessions, leaving the hearing tests and caloric tests usually for the second session, by which time the laboratory work has been completed.

CLASSIFICATION

In order to be of practical value any classification of dizziness must be relatively simple. It has been found advantageous to consider vertigo of vestibular origin in three main groups on the basis of localization. If the case can be classed as belonging to one of these three groups the etiologic diagnosis then becomes somewhat simplified. The various causes can be considered under these three groups.

GROUP 1

This group includes cases in which the vertigo is known to arise from the central nervous system. The localization to the central nervous system is seldom made on the basis of the ear findings alone but is usually based on the associated central nervous system symptoms and signs. Dizziness of central origin is due mainly to involvement of the vestibular nuclei or the directly associated nervous pathways.

In some instances the vertigo may be rapid in onset resembling the vertigo which arises from disease of the peripheral labyrinth. This is true particularly of vascular lesions. In many instances however the vertigo associated with central nervous system disease is postural in character and is brought on when the body and head assume certain positions. The practical importance of postural vertigo is that such information can be readily elicited in the history and the complaint can usually be substantiated by putting the patient through the positions which he has found to cause the symptoms. The reproduction of the vertigo and the observation of a positional nystagmus at that time indicates the existence of an organic lesion of the vestibular system. Some of the central nervous system diseases which commonly produce vertigo are:

1. Inflammatory disease such as encephalitis, meningoencephalitis or brain abscess.
2. Vascular lesions, particularly those affecting the posterior inferior cerebellar artery or related vessels.
3. Tumors, particularly when located in the posterior fossa or affecting posterior fossa structures by pressure or distortion.
4. Degenerative disease, of which the most frequent is multiple sclerosis.

The etiologic diagnosis in the case of the central nervous system lesion is usually dependent upon associated symptoms and signs of central nervous system disease.

In many instances examination of the function of the ear, both as to hearing and vestibular function, will contribute information essential to the localization and determining the cause.

The dizziness which comes with central nervous system disease may require symptomatic treatment if onset is acute and rapid. Complete rest, and the use of drugs such as Dramamine® (meclizine hydrochloride) Bonamine® (dimenhydrinate) and chlorpromazine are of value. In the case of postural vertigo the patient soon learns to avoid the positions that bring on the symptom.

Of practical importance is the fact that a progressive disease such as tumor or multiple sclerosis may cause dizziness, and particularly the postural type of dizziness, as the initial symptom.

GROUP 2

A second group of cases can be localized in origin to the peripheral vestibular mechanism or the 8th nerve. This localization is based principally on the association of auditory disturbances with the onset of the dizziness. While it is true that dizziness

may come from the peripheral vestibular mechanism without tinnitus or deafness, there is usually no way in which this can be established. There are many causes for vertigo arising from the inner ear and 8th nerve. Some of these causes are problems for specialists but a few are of direct interest to general physicians as well.

Labyrinthitis due to spread of infection from the middle ear has been a common cause of severe dizziness in the past but need not enter into the present discussion.

Tumors involving the ear or the 8th nerve, although not common, need to be considered because the early diagnosis of such tumors, particularly acoustic neurinoma and cerebellopontile angle tumors, is essential if cure is to be obtained.

Dizziness may not be a prominent symptom, whereas impaired hearing and tinnitus in one ear usually are the first complaints. A good rule to follow is that every person with unilateral nerve deafness of fairly recent development should have a complete examination both of hearing and of the vestibular function, always with the possibility of tumor in mind.

There are some systemic diseases which occasionally are complicated by a disturbance of the labyrinth. Blood diseases in particular such as severe anemia or leukemia occasionally affect the inner ear. Allergic reactions sometimes produce inner ear disturbance. Certain drugs will also cause impaired hearing and dizziness in susceptible persons. Quinine and its derivatives and salicylates are two of the classic examples.

Of especial interest to medical practitioners are two types of inner ear or vestibular disturbance. One is the sudden or apoplectic attack of dizziness, deafness and tinnitus in a previously healthy ear. There may be one attack in a lifetime. Another is the syndrome of repeated attacks of dizziness associated with fluctuating degrees of deafness and tinnitus which is now known as Meniere's disease.

The sudden attack of dizziness, deafness and tinnitus in the previously healthy ear may occur in any age period from the second decade on. The cause in some cases is infection, in some it is cardiovascular disease and in some it is not known.

Some virus diseases apparently have the capacity to set up a reaction within the inner ear. The best known examples are measles and mumps and some other acute infectious diseases.

The measles virus[2, 3] has been found to set up labyrinthitis in the endolymphatic labyrinth, which injures the sense organs directly. While histopathologic proof has been obtained only in the case of measles, the clinical evidence indicates that some infections of the upper respiratory tract, probably viral in nature, may also affect the labyrinth.

It also seems that the inner ear or 8th nerve may be the site of a toxic reaction to a focus of infection elsewhere—in the paranasal sinuses, for example.

Vascular disturbances in the inner ear are probably the most frequent cause of an isolated sudden attack of dizziness and deafness. Little information is available about the pathologic change in the human ear in such cases, but there is clinical evidence that a vascular accident, probably occlusion, may produce a severe dizzy spell accompanied by varying degrees of permanent damage to both auditory and vestibular function.

Vascular occlusion probably produces a fairly typical clinical picture in that the onset is rapid, recovery is slow, requiring sometimes many months before dizziness is fully cleared up, and there is a varying degree of permanent damage.

A sudden attack of deafness and dizziness without any evident cause sometimes occurs in persons in their twenties and even earlier. Recovery from the dizziness is gradual, hearing may improve but some degree of permanent damage is the general rule.

TREATMENT

Symptomatic treatment includes absolute quiet, sedation and the use of Dramamine, Bonamine, chlorpromazine or related drugs with maintenance of fluid balance by intravenous infusion if necessary. Vasodilator drugs such as nicotinic acid and intravenous histamine or procaine have been tried but with questionable effect. If some function remains there is usually spontaneous improvement in hearing for the first two months.

The syndrome of recurrent attacks of dizziness, along with deafness and tinnitus is a more common condition. It is known as Meniere's disease in most medical writings at present because of the clear-cut clinical syndrome and the evidence that the pathological condition known as idiopathic hydrops of the labyrinth is present in the inner ear in such cases. Meniere's disease is greatly feared by the patient because of the incapacitating spells of dizziness, which are unpredictable as to frequency, duration or severity. The deafness and tinnitus follow a more consistent pattern. The more constant clinical findings are the hearing loss for low tones and the low pitched "roaring" tinnitus, both of which fluctuate in severity from time to time. In about 90 per cent of cases the disease is unilateral. The diagnosis can not be made on the basis of dizzy spells alone.

The cause of Meniere's disease is not yet known. It seems probable that some disturbance in the mechanism of endolymph production must occur and that some alteration in the constitution of the endolymph thereby takes place. The theory cur-

rently favored is that this disturbance in endolymph occurs as a result of autonomic imbalance affecting the blood vessels in the cochlea. There is some clinical evidence that mental or psychic stress as well as nervous fatigue tend to aggravate the condition.

The medical treatment of Meniere's disease has been relatively unsatisfactory. Various medicines have been used with the object of preventing the dizzy spells, including low salt diet, diuretics, vasodilator drugs such as histamine and nicotinic acid and blocking agents such as Banthine and many other drugs. Clinical investigations by Perlman and Goldinger[3, 4] seemed to refute the theory that the disease could be influenced by the salt intake and the use of diuretics.

Routine medical treatment used by the author includes advice to avoid the use of particularly salty foods but not to restrict the use of salt in ordinary quantities in cooking. Nicotinic acid is prescribed routinely, partly because it is simple for the patient to use at home. Fifty milligrams twice a day is the usual dose. So far no convincing evidence has been observed that any of the medical therapy used has had any consistent value. It is important to control the patient's activities, for example, to avoid overwork and to encourage regularity and moderation in habits of eating and resting. A mild barbiturate is usually helpful. It would appear from clinical observation that mental stress and fatigue are important factors to avoid. It is possible that the psychological effect of the general management of the patient may be the most important factor. At least 90 per cent of patients under medical management show definite improvement and may be able to continue normal activities.

During a dizzy spell different methods of treatment have been tried. Intravenous infusions of histamine and of procaine have been used with indefinite results. Procaine block of the cervical sympathetic ganglia have also been tried without definite effect. Dramamine by mouth, or intravenously if necessary, is of definite value. Bed rest and avoidance of movement is essential.

In the past 15 years the author has observed that between 5 and 10 per cent of patients with Meniere's disease have not been sufficiently controlled by ordinary medical therapy and have needed some other type of treatment.

Procaine block of the stellate ganglion has not proven successful in our hands.

Streptomycin in carefully controlled dosage carried to the point where the vestibular responses are abolished has been used in a few selected cases with good results in all. Streptomycin therapy unfortunately gives the patient an ataxia in order to give protection against dizzy spells. This ataxia is soon compensated for in younger patients but less readily in patients in the sixth decade and over. The author is of the opinion that streptomycin therapy is to be considered mainly in bilateral cases in which the dizzy spells are causing definite disability, provided the patient is under about 50 years of age.

Surgical operation to destroy the inner ear is the treatment of choice in unilateral cases in which there is continuing disability from dizzy spells. Complete destruction of the labyrinth is preferable to any attempt at partial destruction. It is also preferable to section the vestibular nerve, for the risk is less and tinnitus is more likely to be relieved.

GROUP 3

In addition to the cases of dizziness which can be localized either to the peripheral or to the central vestibular mechanism, there is a somewhat larger group in which definite localization may not be possible. These are the cases in which patients complain of vertigo but have no deafness or tinnitus and no evidence of any disease of the central nervous system. They may or may not have some associated systemic disease which might suggest an explanation for the dizziness. In this group the patient may have a single attack of dizziness which clears up and does not recur; he may occasionally have repeated attacks or he may have dizziness which is brought on only when he gets into certain positions.

In the event that the patient states that he gets dizzy only when he sits up in the morning, or when he turns on one side in bed, or when he stoops over or looks upward for some moments, it is usually possible to reproduce the dizziness by placing him in these particular positions. These postural tests will confirm the patient's complaint. If the dizziness can be reproduced and is accompanied by nystagmus, it is clear that there is organic vestibular disturbance.

There are a number of conditions that can cause dizzy spells or dizziness of the kind in this group.

Acute infection usually of the virus type frequently causes vertigo either in the form of an attack which may require several days for recovery or in the form of postural vertigo. Occasionally there appear to be mild epidemics of such infections. Disease of this kind has been named "vestibular neuronitis" by Hallpike and associates.

Cardiovascular disease may also cause this type of dizziness. The vertigo which is caused by a vascular accident or occlusion tends to follow a particular pattern. There is an acute onset of vertigo and it remains severe for a few days then gradually recedes. As the symptom decreases it tends to become a postural symptom only. It is characteristic

of this type that several months and even one or two years may be required for complete recovery from the dizziness. The site of the vascular lesion may not be evident. An attack of dizziness of this type in a patient who has definite cardiovascular disease is probably an indication of vascular occlusion. While spontaneous recovery from such an attack is the general rule, it is not uncommon to have other central nervous system vascular accidents.

The treatment consists of bed rest, sedation and the use of Dramamine or a similar drug during the acute stage of the dizziness, followed by the appropriate medical management of the cardiovascular disease.

Dizziness frequently occurs in patients with a hypotensive state and it is usually of postural type. The use of drugs such as ephedrine or ergotrate may be of some value in such patients.

Dizziness is not an uncommon complication at the menopause. Intermittent periods of postural vertigo may occur for one to two years. Hormonal therapy has been used in such cases but with indefinite results. The prognosis is good for complete relief from symptoms.

Certain drugs can cause dizziness and this is frequently a postural symptom only.

Another group of patients who often complain of dizziness are those with injury to the head. A high proportion of persons following a blow to the head may complain of dizziness for several weeks or months. The blow may not be severe enough to cause fracture or even unconsciousness. Usually such patients are dizzy only when they get into certain positions.

The practical importance of postural vertigo is not that it necessarily localizes the lesion, but that if this type of vertigo is indicated by a careful history it is usually possible to reproduce the symptom on postural tests and obtain objective proof by observation of the accompanying nystagmus.

The origin of dizziness following a blow to the head is not certain, but is not of practical importance since the dizziness subsides in time.

There are many cases in which no definite cause of dizziness can be determined. In some cases there may be a single short attack; in others, repeated attacks; and in many cases dizziness may be a purely postural symptom. The latter sometimes lasts for months and does not respond well to treatment. Avoidance of the offending positions is essential.

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